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First Report of the Association Psoriasis and Minimal Change Disease

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ABSTRACT

Psoriasis is an immune-mediated chronic inflammatory disorder of the skin. Association with kidney disease has been debated for a long time. Recently some glomerular diseases have been diagnostic in patients with psoriasis; The underlying pathogenetic mechanisms of these associations remain unclear because of the limited number of cases .We describe the occurrence of a nephrotic syndrome with minimal change disease (MCD) in a 43-year-old patient with a 20 year history of psoriasis .Evolution was favorable under corticosteroids.

Introduction

Psoriasis is a chronic inflammatory disease characterized by the hyper proliferation of keratinocytes, vascular hyperplasia, and mononuclear infiltration of the dermis and epidermis.

This condition affects 0.1 to 2.9% of the general population and represents 5% of all dermatoses [1].

The pathophysiology of psoriasis is not completely elucidated. The extracutaneous manifestations are essentially articular .The prevalence of renal involvement in psoriasis is unknown. The extent of renal involvement in psoriasis is very wide, ranging from positive microalbuminuria to some cases of glomerulopathy [2].

An increasing number of associations between glomerulopathies and psoriasis are being described; however, there is no established link between the two entities [3, 4].

We report a case of the association of psoriasis with nephrotic syndrome .Histopathology diagnostic minimal change disease.

Case Report

Mr X, Y 43 years old, with a history of chronic tobacco use (20 packs-year) and vulgar psoriasis left untreated for 20 years, was admitted for management of a nephrotic syndrome.

No oral steroid therapy, methotrexate, cyclosporin A, or long-term nonsteroidal anti- inflammatory drugs were used since the diagnosis of psoriasis was made.

Upon physical examination, multiple erythematous, hyperkeratotic plaques and papules with silvery squames were observed on the extensor surfaces of elbows, knees, face and scalp (figure 1and 2). There were droplet-like lesions on the lower limbs and buttocks with candle grease sign.

A moderate edema involved the lower part of extremities bilaterally. There were no signs of arthritis on joint examination. The rest of the general physical and systemic examination was noncontributory.

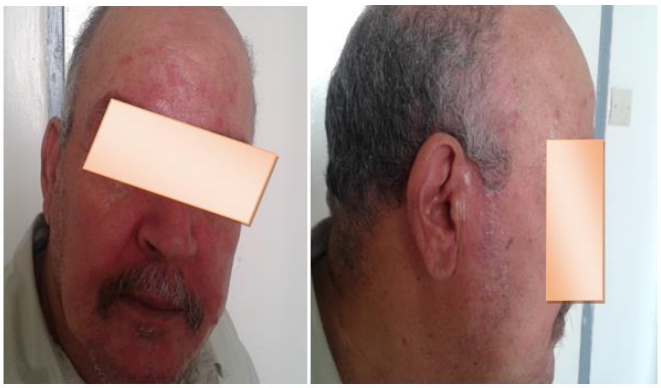

Figure 1 and 2 Erythematous-squamous lesions of psoriasis at the face.

Proteinuria levels were at 5g/24 hours without microscopic hematuria .Renal function was normal (serum creatinine level 0,65mg/dl) with hypoalbuminia (1.0,5 g/dL).

Serum complements C3 and C4 were normal (C3: 86mg/dl, C4: 20,8 mg/dl) .The full blood count and liver function tests were normal (hemoglobin level were 145g/l, ASAT and ALAT levels were respectively 15 and 17 UI/L).

Native anti-nuclear antibodies, Anti neutrophil cytoplasmiq a, anti MBG, were also negative.The hepatitis B and C serologies were negative, as well as HIV, syphilis and toxoplasmosis.

Abdominal ultrasound was normal. Kidneys had normal size and normal index with maintained cortico-medullary differentiation.

Renal biopsy didn't find any cell proliferation or deposit with a fine glomerular basal membrane. (Figure 3 and 4)

The immunofluorescent examination showed negative expression of immunoglobulins IgG, IgA, IgM, complement fragments C3, C1q, and k, l light chains in glomeruli, tubules, and vessels.

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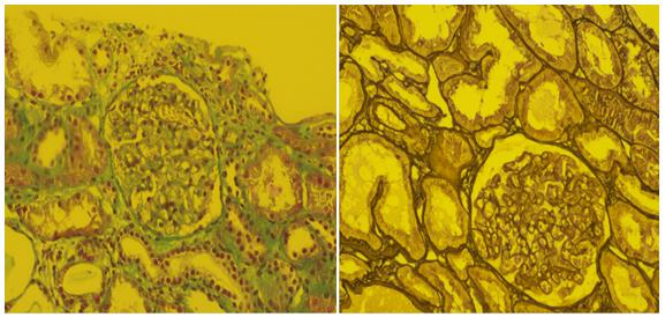

Figure 3. (PAS coloration X20) and Figure 4 (reticuline coloration X 20) , no proliferation or deposits .

Thus diagnostic of nephrotic syndrome with minimal change disease was made.

The patient received conventional oral corticosteroids (1mg/kg/day) with progressive decrease. Total duration of treatment was 6 months.

Follow-up was satisfactory with complete remission of the nephrotic syndrome a month after the start of treatment.

The patient received a local treatment based on emollients for the skin lesions.

Discussion

Psoriasis is an inflammatory disease that affects the skin and sometimes the joints.

Renal involvement in course of psoriasis is not an infrequent event. There are three possibilities for renal association with psoriasis: immune-mediated renal damage, drug-induced renal damage and chronic renal damage [1].

In a recent cross sectional study by Yang and colleagues, renal failure was more prevalent in patients with severe psoriasis than in age and sex matched controls [5].

Various studies have investigated the pathogenesis of kidney damage during psoriasis, leading some authors to describe it as a separate entity called "psoriatic nephropathy" [4].

A high prevalence of urinary abnormalities such as proteinuria and microalbuminuria has been reported in patients with psoriasis [3].

A positive microalbuminuria was found in 22% of patients with psoriasis vulgaris. This rate increased to 42% in patients with severe cutaneous forms [6].

Creamer et al [7] have suggested that increased urinary excretion of albumin is modulated by circulating growth factors such as VEGF and VPF, which are released during skin eruptions.

Glomerular lesions in patients with psoriasis is sparsely reported in the literature: 20 cases of renal amyloidosis secondary to psoriatic arthritis [8] and 10 cases of IgA nephropathy [9].

High levels of Immunoglobulin A and circulating immune complexes have also been found in patients with psoriasis [10].

Patients with psoriasis have a structural defect of immunoglobulin A in 25% of cases, leading to their deposition at the mesangium.

This may reflect a general hyperactive response of the immune system to a hypothetical infectious agent [10].

As in IgA nephropathy, a genetic predisposition seems to exist in patients with psoriasis.

Despite these elements no causal link between psoriasis and IgA nephropathy has been demonstrated [9,11].

In 2009 Akoglu et al. [12] reported the association of psoriasis with membranous glomerulonephritis in an 18-year-old patient without any secondary causes being found.

Other types of kidney damage during psoriasis are secondary to drugs used in the treatment of the condition.

Nonsteroidal anti-inflammatory drugs are responsible for lesions of interstitial nephritis.

The nephrotoxicity of cyclosporine is secondary to a decrease in renal perfusion due to its vasoconstrictive effects while methotrexate causes lesions by its precipitation in the tubular epithelium [13].

Biotherapies have become a treatment of moderate to severe or resistant (to conventional psoriasis treatments) forms.

These are essentially anti-TNF α molecules (infliximab, adalimumab, and etanercept).

A series of clinical cases of glomerulopathies that occurred during treatment were described [14].

In our case report a nephrotic syndrome with minimal change disease has been associated with psoriasis in a patient who has never been treated with cyclosporine or méthotrexate or biotherapies.

This would be the first report in the literature on the occurrence of a nephrotic syndrome with MCD in a patient with psoriasis. Evolution was favorable under corticosteroids.

Conclusion

Our study reports a case of MCD nephrotic syndrome associated with psoriasis. To the best of our knowledge, this is the first publication concerning the association of MCG nephrotic syndrome and psoriasis described in the literature.

Although there is no causal relationship between psoriasis and glomerulopathy outside of amyloidosis, the understanding of renal abnormalities during psoriasis is still imperfect, hence the need for further trials and clinical studies.

The authors report no conflicts of interest.

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